the growth was found quite immovable and apparently firmly attached to the posterior wall of the pharynx. The finger, introduced through an incision between the tumor and the mucous membrane covering it, completely enucleated the growth, leaving its entire membranous envelope intact. The patient made a good recovery. A number of somewhat similar instances were reported by gentlemen who took part in the discussion of this paper before the Section on Surgery and Anatomy of the American Medical Association at its fifty-third annual meeting.

Foreign Bodies in the Air Passages; Escape of the Outer Tube of a Durham Tracheal Canula.—DR. E. D. FERGUSON, of Troy, N. Y., reports (*New York State Journal of Medicine*, October, 1902) the escape into the right bronchus of the outer tube of a Durham double canula in consequence of the neglect of the manufacturer to provide it with the necessary protective flange at its outer extremity. Its removal was effected by incising the tracheal wall down toward the bifurcation, when, with the finger, the missing tube was felt in the right bronchus, the upper extremity being well within the bronchus. With the aid of illumination from a head mirror the upper edge of the tube was seen, and it was removed with forceps.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF
SIMON FLEXNER, M.D.,
PROFESSOR OF PATHOLOGY, UNIVERSITY OF PENNSYLVANIA.

ASSISTED BY
WARFIELD T. LONGCOPE, M.D.,
RESIDENT PATHOLOGIST, PENNSYLVANIA HOSPITAL,

AND
F. P. GAY, M.D.,
ASSISTANT DEMONSTRATOR OF PATHOLOGY, UNIVERSITY OF PENNSYLVANIA.

Multiple Progressive Hyaloserositis (*Studies from the Royal Victoria Hospital*, Montreal, vol. i., No. 3).—Under the foregoing title NICHOLS discusses the etiology, morbid anatomy, and clinical aspects of that form of a chronic proliferative process affecting serous surfaces "characterized by the formation of a membrane composed of dense layers of connective tissue of a peculiar cartilaginous appearance." This membrane, which may be several centimetres in thickness, is a continuous stratified layer, and can be peeled off from the subjacent affected organs. The upper part of the peritoneum, particularly the liver and spleen capsules, the bases of the pleural cavities, and the pericardium are the parts oftenest involved. As a result, the structures attacked may show atrophic, degenerative, or even fibroid changes.

As a localized liver lesion the process has been described as chronic hyperplastic perihepatitis, chronic deforming perihepatitis, and by Curschmann as the "zuckerguss" or iced liver, from the porcelain-like or "frosted" appearance. Though the liver may be primarily and solely involved, the process is usually progressive, affecting the serous surfaces consecutively, but all may not show the peculiar "icing" appearance. The main theories as to the causation of the process are pericarditic pseudocirrhosis (Pick), chronic nephritis (Hale White), and inflammation (Curschmann).

From a careful study of his own and thirteen reported cases of "zuckerguss" liver, Nichols would prove that the first two hypotheses are incorrect. Reasoning from the clinical and pathological evidences at hand, he concludes that the etiological factor is a chronic inflammatory process caused by some micro-organism of slight virulence. The tubercle bacillus is capable of producing quite similar but still distinct lesions.

The pathological findings make clear many of the clinical manifestations. Ascites is constant; anasarca and emaciation are common. The great omentum is often contracted and indurated. The liver, which is usually almost completely invested by the frosted membrane, is generally small and deformed, with round edges and a smooth surface. In the great majority of cases there is no cirrhosis; there are, however, fatty and parenchymatous changes and passive congestion. The gall-bladder is frequently involved in the perihepatitis. The right pleural cavity is more frequently seriously involved than the left. This is strikingly more common at the base of the right lung, the central tendon of the diaphragm, and the right side of the pericardium. Chronic adhesive pericarditis is usually present; the pulmonary and cardiac changes are what would be expected under the conditions. The spleen, which is commonly enlarged, is often frosted, particularly when there is left-sided pleural involvement. It may be atrophic in advanced cases. No special abnormality appears in the kidneys. An acute terminal infection of many of the serous sacs or of the lungs is frequent.

Histologically, this "icing" is found to be a laminated fibrous tissue, with more or less hyaline transformation and giving evidence of being an organized inflammatory exudate.

Clinically, only the cases with the frosted liver are found to admit of diagnosis, and the symptoms vary considerably according as the liver is primarily or secondarily affected.

The average age of onset is about thirty years; the duration varies from two to sixteen years, averaging six years. The sexes appear to be about equally affected. The onset may be either acute or very insidious. The primary infection is most frequently in the pericardium, next in or about the liver, and less often some other peritoneal inflammation. Alcohol, gout, and syphilis appear to have little and heredity absolutely no etiological significance. The constant and striking physical sign is the extreme ascites requiring repeated paracentesis. The ascitic fluid is straw-colored, of a specific gravity of less than 1010, but twice as rich in albumin as a transudate, which is cited as evidence of its inflammatory causation.

If the process is primarily pericarditic anasarca appears first, then a train of symptoms following the extension of the inflammatory process to the liver *via* the right pleural cavity, and finally the ascites. If the liver is primarily involved there is epigastric pain, sense of fulness in the abdomen, and dyspnœa, followed by a slowly developing ascites, and then perhaps an extension to the right pleura and pericardium, with consequent clinical signs and symptoms. Icterus is not present in uncomplicated cases. The urine is decreased in amount, and albumin is commonly absent. The prognosis is eventually hopeless. The treatment is purely symptomatic. Unusual precaution should be taken against exposure to acute infections.—J. L. Y.

The Intestinal Origin of the So-called "Fever of Infancy" (Fiebre de Borras).—Duenas (*Revista medica Cubana*, January, 1903, No. 1) suggests the possible relation of this Cuban infantile fever with the summer diarrhœa of infants of the United States. The recent work of Duval and Bassett has shown that at least certain forms of summer diarrhœa of children are due to the bacillus dysenteriæ of Shiga, as shown by the presence of this organism in the stools of such cases and the agglutination reactions on the organism with the blood of those ill of or convalescent from the disease.

In a case of the author's, a girl of twenty months, who had been suffering for several days with persistent bloody vomiting, pain in the epigastrium, and mucus and ill-smelling stools, a distinct fever and rapid pulse, the patient's blood twenty days after the beginning of the disease gave a positive reaction with the bacillus dysenteriæ in dilutions of 1:60. This case the author further regards as significant of the possible relation of this bacillus with certain of the "black vomits" attended with fever and of unknown etiology.—F. P. G.

The Inheritance and Intra-uterine Transmission of the Agglutinating Properties of the Blood and the Formation of Agglutinins in the Bodies of Embryos.—Jurewitsch (*Cent. f. Bac. u. Para.*, 1902, Bd. xxxiii. p. 76) states that the agglutinating properties of the blood of normal pregnant rabbits and guinea-pigs for typhoid bacilli was first determined. The blood of some normal rabbits possessed this power in 1:80 dilutions. The blood of the young from such mothers also had the power to agglutinate typhoid bacilli, but in less degree than the mothers. Occasionally in a litter from a single mother certain animals showed agglutinins in their blood, while others did not. With normal guinea-pigs neither the blood of mothers nor young showed any tendency to agglutinate typhoid bacilli. Normal pregnant guinea-pigs were now immunized against typhoid bacilli during the time of pregnancy, so that at the birth of the young agglutination took place with the mother's blood in 1:4000 or 1:5000 dilutions. In three out of thirty-one cases no agglutination could be demonstrated in the young. In all the other cases agglutination was obtained with the blood of the young, but was three to thirty times weaker than with the blood of the mothers, and after a short period the blood of the young lost this property entirely. In order to determine whether the agglutinins were simply transmitted through the placenta to the blood of the young, or whether the fœtus was actually endowed with the power of producing agglutinins, the mothers were subjected to a passive immunity, and agglutinating sera from previously immunized guinea-pigs, rabbits, and horses were injected into the pregnant mothers. After a few hours Cæsarean section was performed and the blood of the young tested. The result was the same as when the mothers were immunized during pregnancy. It was further found that the blood of young normal rabbits coming from a